

Campbell (F. M. H.) + 20
L54

ANGINA PECTORIS.

BY

FRANCIS WAYLAND CAMPBELL, M.D.,

LICENTIATE OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON, ENGLAND; LICENTIATE OF
THE COLLEGE OF PHYSICIANS AND SURGEONS, LOWER CANADA; MEMBER OF THE
ROYAL MEDICAL SOCIETY OF EDINBURGH; CORRESPONDING MEMBER
OF THE DUBLIN MICROSCOPIC CLUB, &C., &C.

280,

Re-printed from the British American Journal.

MONTREAL :

PRINTED BY JOHN LOVELL, ST. NICHOLAS STREET.

1862.

ANGINA PECTORIS.

BY

FRANCIS WAYLAND CAMPBELL, M.D.,

LICENTIATE OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON, ENGLAND; LICENTIATE OF
THE COLLEGE OF PHYSICIANS AND SURGEONS, LOWER CANADA; MEMBER OF THE
ROYAL MEDICAL SOCIETY OF EDINBURGH; CORRESPONDING MEMBER
OF THE DUBLIN MICROSCOPIC CLUB, &C., &C.

*(A Paper read before the Royal Medical Society of Edinburgh, on the
28th March, 1861.)*

MONTREAL:

PRINTED BY JOHN LOVELL, ST. NICHOLAS STREET.
1862.

ANGINA PECTORIS.

Mr. President and Gentlemen :

In attempting this evening to draw your attention for a few moments to a disease of such great importance as "Angina Pectoris," I do so more in the hope that on the conclusion of my remarks, the members of this Society will freely state what has been their experience, than of adducing anything particularly new or startling concerning an affection, the pathology of which is still disputed by the most eminent authorities of the day. Fortunately for the human family this disease is of comparatively rare occurrence—yet, it was my good fortune that the first case of importance that I was called to attend, after my graduation, was a genuine case of "Angina Pectoris." As it will form the subject of a portion of the following remarks, I will now proceed to detail it:

On the 4th of July, 1860, I was sent for in great haste to visit James S—, a colourer and whitewasher, aged 54, who, I was informed, shortly after taking a hearty dinner, was seized with a violent pain in the region of the stomach. When I arrived at his dwelling I found the pain had entirely disappeared, and he was comparatively well. His bowels being torpid, I ordered ten grains of blue pill to be taken at bed time and a seidlitz powder in the morning; and left instructions, should the pain trouble him again, to apply a sinapism over the affected region. On the following day, I again visited him, and was informed that twice during the night he had had a paroxysm of pain which the sinapisms failed to relieve. Having made minute enquiries into the man's habits, I found that he indulged rather freely in liquor; this, with the fact that the two attacks he suffered from during the night, were accompanied by a desire to vomit, led me to order a blister over the epigastrium. On the 6th he said he was rather better, the blister having lessened the intensity and frequency of the paroxysms.

On the 7th he was, to use his own expression, "much worse," having had this morning two severe paroxysms. This time he referred the seat of pain to the region of the heart, and described it as agony the most intense, rendering

him almost unable to breathe. It came on suddenly, shooting to the back of the neck, then down both arms, lasting about twenty minutes, and gradually passing off. Clearly now I had a case of "Angina Pectoris." On examining the cardiac region, I detected on close attention a faint and almost imperceptible murmur with the first sound of the heart. I prescribed gr. xv of Dover's powder every four hours, and a table spoonful of the following mixture every two hours: G. Spt. Eth. Sulph. Co., Spt. Amon Arom $\bar{a} \bar{a}$ $\frac{2}{3}$ ss. Tinct. Hyoscyam 3 iii, Aquæ ad $\frac{2}{3}$ vi. On the morning of the 8th instant he walked to my surgery, and asked me to repeat the powders, as they had done him a great amount of good. Had during the previous night only one paroxysm, which was mild in character compared to those which preceded. On the 9th of July, I visited him about noon; he felt himself improving, and was in much better spirits; wished to go out to attend to some business which I forbade. Same medicines continued. About half past 7 o'clock, contrary to instructions, he attempted to cross the street to a neighbour's house, when he was seized with a severer paroxysm than any of the preceding, so much so, that he was unable to return home without assistance. As he was thought to be dying, I was sent for in great haste. I found him trembling violently, and his body covered with cold perspiration; the pulse was small and frequent, and the countenance exhibited extreme anxiety. A little brandy was ordered, otherwise to continue as before. About 9 o'clock I saw him with Dr. Craik in consultation, when matters were found much as reported on the 7th. While we were in the room, a paroxysm came on, and on placing the stethoscope over the heart, a loud systolic murmur was heard, also a very distinct and harsh dactylic bruit; both these sounds were heard over the aortic valves but were inaudible at the apex. All previous medicines stopped, and the following ordered: R. Tinct. Valerian Amon 3 vi, Spt. Eth. Sulph. Co. $\frac{2}{3}$ ss., Tinct. opii 3 ii., Aquæ ad $\frac{2}{3}$ vi., take $\frac{2}{3}$ ss., every three hours. It was determined to try the hypodermic injection, should we again find him in a paroxysm. On the 10th he felt easier, and a few moments before I arrived on the 11th, he was attacked with a paroxysm, which was on when I entered. The physical signs were precisely as noted on the 9th. I injected vii gtt. of Tildens Fluid Extract of Belladonna, and xx gtt. of Liquor Opii Sedativus (Battles) hypodermically, which failed to give any relief or affect him in the slightest, and the paroxysm passed off as previously.

12th July. Dr. Craik met me in consultation to day. Has had several severe attacks since last visit. We felt inclined again to try the effect of the hypodermic injection, but he positively refused to submit; he begged to be cupped which I accordingly did, and obtained about six ounces of blood. 13th July. Has found more relief from the cupping than from any thing else. Asked to have it repeated which was accordingly done. On the following day he felt himself so much better, that it was with difficulty he could be persuaded to remain at home. 15th July. On making my visit to day, I found that in the morning he had had a slight attack, which he attributed to some exertion he had made. Visited him at 8 P. M.; felt quite well, was sitting chatting with some friends. No paroxysm since the morning. 16th July. About three o'clock this morning, he suddenly awoke his wife saying he had another attack. He

then called for his medicine. She got up, lit a candle, despatched a messenger for me, and brought the bottle to him. He raised his head to take some of it, when he suddenly placed his hand over his heart, his head fell back, and without a struggle he died.

AUTOPSY.—A *post mortem* examination was made at four o'clock this afternoon, being about thirteen hours after death. The countenance was tranquil and the cadaveric rigidity extreme. The heart was considerably enlarged and fatty. On the surface were two milky patches, about an inch in diameter, and each of the cavities contained a small quantity of blood. The mitral and tricuspid valves were healthy. On the free surface of the aortic valves, ossific matter was deposited, as well as upon the whole surface of the arch, rendering the parts rough and gritty to the finger. At the aorta between two of the valves, was a triangular spot, about three eighths of an inch in diameter, which projected to the extent of one tenth of an inch into the calibre of the artery, and no doubt contributed to cause the murmur which had been diagnosed before death. No other lesion of the heart was discovered, and all the other organs were healthy, with the exception of the liver, which, as might have been anticipated, by the man's habits, was considerably enlarged."

This, gentlemen, terminates the history of a case, which to me was full of interest. Doubtless it is not as fully reported as one more experienced would have done, still I hope that in some points at least it is instructive. As a general rule "*Angina Pectoris*" does not terminate fatally so rapidly, as in the case just detailed, for Stokes records a case where the patient suffered for ten years from aggravated symptoms of this disease. Indeed, so far as time and the means at my command allowed, I have been able to find but one recorded case which proved fatal in a shorter period, which is given by Latham, and was that of Dr. Arnold, the head master of the School at Rugby. Others may have occurred—may be reported—but they can be but few, for Dr. Begbie of this city, Professor of Practice of Medicine in the College of Surgeons, whose means of enquiring are of course great, a few weeks ago while lecturing on "*Angina Pectoris*," was pleased to quote my case, as an example of an extremely rapid termination of the disease. Previous to my being called to attend this man, he had enjoyed remarkably good health, for a period of twenty years not having a bodily ache of any kind. The quick succession of attacks which he suffered from was another peculiarity in the case, while no exciting cause could possibly be ascertained. In almost all the cases which I have read, weeks and months as a rule intervened between the paroxysms; while my patient had three and four in one day, the fatal termination ensuing upon the twelfth day from the first attack. It is deeply to be regretted that concerning a disease so interesting as the one under consideration, that of late years but little has been done towards its investigation. In such works as Walshe, Stokes, and Latham, we find that a few pages contain all these justly celebrated authors have to say on the subject. It is, I say, to be regretted; for of late years the microscope has thrown such a vast amount of light upon hitherto obscure affections, that I cannot resist the temptation of believing that if those who from their extensive field of observation, are likely to have cases of "*Angina Pectoris*," come under their

notice, would patiently investigate the subject with the aid of that valuable instrument, that the darkness and uncertainty which now surrounds it, would soon pass away. As it is now, we have to go back to the year 1799, since which time but little advance has been made in our knowledge of this disease. In that year Dr. Parry, a member of this Society, published a work, entitled "An enquiry into the symptoms and causes of Scyncope Anginosa," and to this day, it is most unquestionably the best monograph that we possess on the subject, and the theory then advanced by him I will presently attempt to show is the one most supported by pathological observation. One fact concerning the disease we may however take as established, and that is true "Angina" never occurs without organic disease of the heart or arteries in its vicinity. It is true, cases are recorded in which no traces of organic disease were observed. Concerning such cases Dr. Stokes (and I can but believe he is correct) says: "It is more probable that in the cases so described, the disease was overlooked: than that the heart was perfectly sound." He then goes on to say, "that such cases as were observed before the application of the microscope to pathological anatomy may be set aside, as proving the existence of "Angina" without organic change; for among the most important uses of histological research, is the discovery of those early stages of organic change, which escape the unassisted eye." Dr. Walshe on the same subject says, "It has occurred to me to examine during life some six or eight cases of true "Angina"; in every one there were signs of organic disease. I have opened or seen opened the bodies of three persons destroyed in the paroxysms; the heart was texturally affected in all." The form of organic disease present as enumerated by Latham are as follows:—1st. Weakness and attenuation; 2nd. Weakness with fatty degeneration; 3rd. Some form of valvular disease, generally affecting the left side; 4th. Disease of the aorta with or without obstruction of the coronary arteries. If we analyze closely the various cases which have been recorded it will be found that in the great majority of instances, the organic disease present was weakness with attenuation of the walls of the heart or weakness with fatty degeneration, the coronary arteries as a rule being ossified (and as in the case detailed, the ossification extending frequently to the aorta and valves) and if not truly in an ossified condition, at least a cartilagenous formation being found in their interior. If the coronary arteries are found in the condition I have just named, it need not I think excite our amazement, if we find the heart itself in a weakened condition; for just in proportion as these arteries vary from their normal state will the nutrition of the heart be impaired. If the calibre of the artery is in the slightest degree diminished, the required amount of blood will not reach the great arterial centre, the result being a weakening of the muscular fibres. I believe, then, that as a general rule in all true cases of "Angina Pectoris," the coronary arteries will be found diseased. Having made this strong assertion, it may surprise the members of this Society somewhat, that in the case which came under my care not one word about the coronary arteries appears in the post mortem examination. You cannot regret it more than I do, but it could not be avoided. It was with great difficulty that I succeeded in getting the friends to consent to such an examination, and before it was completed to our satisfaction, the friends

entered, and would not allow us to proceed further. Unfortunately the examination of the coronary arteries had been postponed till the last, but from hurriedly passing my finger over them, I can with certainty say, they felt as if ossified, and in my own mind I have no doubt but they were.

It will be remembered that the heart of this man was enlarged and fatty. The hypertrophy can easily be accounted for from the man's occupation, which was of such a character as to constantly keep the heart in an excited condition. It is very probable that he may have suffered, unknowingly, from this disease for many years; the fatty condition, judging from its extent, being of comparatively recent date. My opinion is, that had the man lived a few years longer, the hypertrophy would have in a great measure disappeared; while the muscular fibres of the heart to the naked eye—but more so under the field of the microscope—would have presented in a very marked degree the characteristic signs of weakness, the fatty transformation in the meantime gradually increasing. I have previously mentioned a case, recorded by Stokes, of a patient suffering ten years from "Angina Pectoris," and here the *post mortem* revealed vast hypertrophy, which was believed to have been secondary to an attack of "Endo pericarditis." I regret that in this case not a word is said regarding the condition of the coronary arteries; still, however, I am forced to believe they must have been in an abnormal condition, sufficient to interfere with the proper nutrition of the heart, for I find that *Digitalis*—a most valuable remedy in the treatment of hypertrophy—was sure to aggravate his distress; saline purgatives did the same. Now surely, if the hypertrophied heart was in this case, as is the rule, receiving an amount of blood equal to the increased duty which it was called upon to perform, *digitalis* would have been the remedy above all others selected to benefit the patient. On the contrary, he always received the greatest possible benefit from the employment of stimulants, as a rule contra-indicated in hypertrophy, his allowance being for many years eighteen tumblers of punch daily. The effects of these two directly opposite modes of treatment goes strongly, I think, to prove that the muscular fibres of the heart were in an excessively weakened condition. Hypertrophy in "Angina Pectoris" seems to be extremely rare. In addition to my own case, the only other I have been able to lay my hands upon (having used your magnificent library) is the one I have quoted from Dr. Stokes, and if we will not admit that the fibres are degenerated, it will be somewhat of a difficult task to account for the sudden death of Dr. Stokes's patient, which he himself says was by syncope. Before taking leave of this case, I may as well mention that none of the salts of morphia, or even the black drop, except in *very large* doses, produced the desired effect.

Having thus touched upon a few of the leading facts connected with this disease, we will now pass on to consider the real and important question at issue, What is "Angina Pectoris"? I have mentioned the different varieties of organic lesion which are found in and around the heart; but as all these changes exist and prove fatal without any symptoms of "Angina," "the conclusion is unavoidable," says Walshe, "that there is something beyond organic mischief concerned in generating the paroxysm." What that is, I will now try to show. I may fail; if I do so, I fail in a good cause. Walshe, with Latham and others,

allies it to the nervous group, stating it to be spasm of the heart; while Stokes, with Parry, who is still the great authority on this disease, considers the pathological condition during the paroxysm to be a diminution of the muscular power of the heart: and from what I have already stated, you know that this is the theory which to my mind seems to be most strongly supported by facts. First, however, we will see what arguments the supporters of the spasmodic theory have to adduce. Walshe says that its spasmodic character appears from its sudden advent and departure; from the character and intensity of the suffering; from the perfect ease enjoyed in the interval of seizure; and from the kind of treatment that proves beneficial. Latham urges more strongly than the preceding authority its spasmodic character. He refers the pain, and the dying sensation felt by the patient, to spasm. Certainly these arguments appear strong, but, if we have recourse to facts, we find that the theory advanced by Parry, and more recently endorsed by Stokes,—that the attack depends upon a weakened condition of the heart, the blood arriving at that organ faster than it is able to propel it onward,—is the one most supported by them. Thus females, notoriously more liable to diseases of a spasmodic character than males, enjoy almost a complete immunity from it: indeed, I consider it very doubtful if the few cases that are mentioned as occurring in women were genuine attacks of Angina Pectoris. It nearly always occurs in men above the age of fifty (a period of life peculiarly prone to ossification of the arteries) and of a leuco-phlegmatic habit of body, and there is not the slightest evidence to show that after a fatal paroxysm of Angina the heart has been found in a spasmodically contracted condition, although this condition has been found after death from other causes, such as tetanus or decapitation. In my own case, although the muscular system generally was in a state of extreme rigidity, the heart, so far from being spasmodically contracted, was more flaccid than usual and contained blood in all its cavities. The walls of the organ were attenuated, and in a state of fatty degeneration, two conditions not at all favorable to spasm, but eminently so to failure of muscular power. If Angina was due to spasm, should we not expect to find, and *should we not* find the heart firmly contracted? Again, if it was spasm, would not the circulation be interrupted, in fact totally stopped, which we certainly know is not the case. On the contrary, we find that the pulse becomes weak the moment the paroxysm sets in, and increases in weakness just in proportion to the duration of the attack. Dr. Parry, in his admirable work, mentions the case of a patient who was under his care for a lengthened period, who permitted the Doctor to accompany him during a walk up hill, in order that he might witness what occurred during a paroxysm of Angina. Dr. Parry says, “when the fit was thus excited, I could perceive no symptoms of disorder in addition to the uneasiness at the breast, except a *gradual* and most *evident diminution* of the strength of the pulse, and I have no doubt that we shall invariably find the pulse become weaker in proportion to the intensity of the paroxysm.” Dr. Wall also mentions a case where the pulse was never *irregular*, but *always small*, gradually sinking as the paroxysm increased. Can we account for this state of things on the theory of spasm? I think not; yet it seems to me no difficult matter to account for them if we only admit the weakened condition of the

muscular fibres of the heart. The cold perspiration, which is so frequently seen in a paroxysm of any duration, is easily explained by the gradual failure of the circulation, and the intense anxiety of mind under which the patient labors. Many patients complain that, when in the paroxysm, they have the terrible feeling that to take a full inspiration would cause instantaneous death, yet that it is only a feeling is proved by a case mentioned by Parry. A gentleman had this terrible feeling, yet when he could muster sufficient courage to take a deep and full inspiration, he found the *greatest possible relief* from it. May we not account for this by the expanding lung pressing against the gradually distending heart, and thus assisting it in its getting rid of the accumulating blood. Taking a deep inspiration, and retaining the breath, affords relief from this feeling so long as the lung is expanded. This can, I think, be accounted for in the same way. Added to the weakened condition of the heart, is, as I have said before, often found ossification of the large vessels or valves about the heart, which will prevent the free evacuation of the blood from the cavities, and in this way assist in dilating them; and I think we can easily understand how the elasticity or living force of an organ like the heart may be overcome by extreme distension. Hence, though a heart diseased may be fit for the purposes of common circulation during a state of bodily and mental tranquility, and of health otherwise good, and yet when any unusual exertion is required its powers may fail under the new demand, accordingly we find that "Angina" is readily excited by those passions the tendency of which is to stimulate the heart to excessive contraction. Thus we find that many of the recorded cases proved fatal while the patient was in a violent transport of anger.

The symptoms of Angina are as readily accounted for on this theory, for the pain may be due to the distension of the heart and large veins; indeed the increased volume of the heart more readily accounts for the pain shooting along the arms from pressure upon the nerves, than from any phenomena connected with spasm. Mr. Home, however, attributes the pain to the pressure of the nerves of the heart against the rigid coronary arteries during the paroxysm. The cause of sudden death from "Angina Pectoris" while the patient is asleep, can hardly be accounted for in the same way as I have accounted for a paroxysm. It is more likely, I think, that the patient suddenly starting in his sleep, the blood is driven forward, to a heart already in a very weak condition, with such an impetus that the violent effort it makes to contract, and propel the blood onward, is too much for some of its attenuated fibres, and that a minute rupture takes place, causing instantaneous death; the rupture being so minute as to be unobservable to the naked eye. Such is the opinion I have formed regarding sudden death from Angina while the patient is asleep. Before closing, I will notice one objection, or rather one more argument, which the supporters of the spasmodic theory may be inclined to adduce. They may say that the spasm may be only sufficient to impede, not destroy the circulation. To this Stokes says it is difficult to understand how such a thing should occur, for a complete spasmodic closure of any one cavity ought to cause death by breaking the continuity of the circulation. As regards the use of opium, it seems to be of but little value, except as a narcotic; if it was likely to benefit as an anti-spasmodic,

surely it would have been of use to my patient when injected hypodermically (at almost the commencement of a paroxysm), the most speedy way of its entering the circulation.

The difference of opinion respecting the condition of the heart during a paroxysm might be overlooked, were it not likely to influence the treatment; but as the treatment of spasm differs materially from that of debility, the disputed point becomes one of some importance. If, for instance, the spasmodic view be adopted, the great remedy ought to be found in chloroform; but I have been unable to find any recorded cases in which its administration has been attended with benefit. On the contrary, Dr. Stokes mentions a case of *intercostal neuralgia*, in which the external application of chloroform produced convulsions and collapse, which lasted for many hours. Adopting the other view of the pathology of the disease, the remedies which should be used ought to be stimulants, together with the careful avoidance of all depressing agencies, such as over-exertion of mind and body, ebullitions of temper, sudden surprises, &c., &c. The treatment of course only can be palliative, for the constant concurrence of organic disease precludes the possibility of a permanent cure.

Before closing, I can but express the hope that the next few years may elicit more concerning the pathology of this interesting disease than has the previous sixty-three years.

On the conclusion of the paper an animated discussion took place, which lasted for about two hours, in which Mr. Pettigrew, whose dissections of the heart elicited so much admiration, Mr. Berryman, assistant to Professor Simpson, Dr. Capie, Mr. Duncan, Mr. Crichton Brown, and numerous others took part,—the majority speaking against the spasmodic theory.

